	1994-95	1996-97	1998-99	2000-01	2002-03	2004-05
	N (%)	N (%)	N (%)	N (%)	N (%)	N (%)
# of patients						
Total	15,603	19,765	20,260	15,989	16,125	18,244
AutoHCT	10,557 (68)	13,628 (69)	13,931 (69)	9,602 (60)	9,449 (59)	10,918 (60)
AlloHCT	5,046 (32)	6,137 (31)	6,329 (31)	6,387 (40)	6,676 (41)	7,326 (40)
Median (range) Age, years						
AutoHCT	44 (<1-75)	46 (<1-76)	48 (<1-78)	50 (<1-78)	52 (<1-91)	53(<1-81)
AlloHCT	33 (<1-68)	35 (<1-79)	37 (<1-76)	39 (<1-79)	40 (<1-76)	40 (<1-78)
AutoHCT graft source						
Marrow	2,730 (26)	1,470 (11)	651 (5)	296 (3)	193 (2)	208 (2)
Blood	6,288 (60)	11,216 (82)	12,773 (92)	9,020 (94)	9,104 (96)	10,618 (97)
Marrow + Blood	1,539 (15)	942 (7)	507 (4)	286 (3)	151 (2)	92 (1)
AlloHCT graft source						
Marrow	4,571 (91)	4,693 (76)	4,420 (67)	2,834 (46)	2,111 (32)	1970 (27)
Peripheral Blood	357 (7)	1,129 (18)	1,640 (26)	2,996 (47)	3,912 (59)	4,588 (63)
Marrow + Blood	33 (1)	36 (1)	45 (1)	26 (<1)	11 (<1)	27 (<1)
Cord Blood	85 (2)	279 (5)	404 (6)	531 (8)	642 (10)	741 (10)
AlloHCT donor relationship						
HLA-identical Sibling	2,999 (59)	3,555 (58)	3,547 (56)	3,443 (54)	3,400 (51)	3,255 (44)
Unrelated	1,237 (25)	1,802 (29)	2,024 (32)	2,182 (34)	2.616 (39)	3,273 (45)
Other Relative	614 (12)	623 (10)	575 (9)	590 (9)	496 (7)	525 (7)
Other	25 (1)	18 (<1)	19 (<1)	61 (1)	87 (1)	160 (2)
Missing	171 (3)	139 (2)	164 (3)	111 (2)	77 (1)	113 (2)

declined by >30% in the early 2000s, but rose again in 2004-5. Most of this change is due to a decrease in AutoHCT for breast cancer from 42% in 1994-5 to 1% in 2004-5. In this same period, AutoHCT increased for MM (6% to 44%) and NHL/HL (33% to 41%), whereas acute or chronic leukemia decreased from 8% to 5% and solid tumors remained stable at 9%. The number of AutoHCTs for the new indication of autoimmune diseases have totaled 146 from 1996-2005. The number of AlloHCTs increased by 45% over this 12-year period, mostly due to a 2.6-fold increase in unrelated donor AlloHCTs. From 1994-5 to 2004-5, AlloHCT increased for AML/ALL (43% to 51%), MDS/MPS (8% to 11%), and NHL/HL (8% to 12%), but decreased for CML (23% to 8%) and MM (5% to 1%). AlloHCT conditioning regimen intensity was 100% myeloablative in 1994-5, whereas reduced intensity/nonmyeloablative regimens were used in 28% of AlloHCTs in 2004-5. The use of peripheral blood and cord blood have dramatically increased. The median age and upper age limit have increased for both AutoHCT and AlloHCT. The percent of non-White race recipients increased from 1994-5 to 2004-5 for both autoHCT (8 to 17%) and AlloHCT (14 to 19%). In the entire study period, there have been 2,115 planned tandem AutoHCTs, 242 planned tandem Auto/AlloHCTs, 48 planned tandem Allo/AlloHCTs, 1,135 unplanned Auto/AutoHCTs, 1,559 unplanned Auto/AlloHCTs, 2,091 unplanned allo/AlloHCTs, and 2,828 other multiple HCTs (unknown if planned or unplanned). The HCT population has changed dramatically over time. Further analyses are ongoing to determine the impact of these changes on the success of HCT.

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AUTOLOGOUS STEM CELL TRANSPLANTATION (ASCT) FOR HIV-RELATED LYMPHOMAS IN THE HAART ERA: A META-ANALYSIS OF RESPONSE AND SURVIVAL POST TRANSPLANT

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Introduction: Recently data has shown survival and response rates in small numbers of HIV patients receiving ASCT for HL and NHL in the HAART era. We did a comprehensive review and meta-analysis of the literature to examine collective therapeutic outcomes and survival.

Methods: We searched OVID, PUBMED, Google Scholar (1980 – Sept 2009), ASH (2004-8), ASCO (2004-9), and BMT Tandem (2005-9) annual meeting abstracts for references. Articles obtained were reviewed for additional references. Inclusion criteria: 1) Autologous transplants for HIV/AIDS associated lymphoma with HAART 2) Reported in English 3) Studies reporting: complete response rates (CR), and one or more survival statistics. 4) Comprehensive trials, cohort studies or case series. Authors were queried for possible patient overlap among studies. Primary outcome measures were response to ASCT and 2 year overall survival rates (OS).

Results: Out of 61 references, 14 studies were on ASCT in HIV related lymphoma and 13 case reports. 5 met inclusion criteria. These included 35 Hodgkins and 83 NHL subjects. CR rates post ASCT 71% (95%CI 60-80), 2 year OS of 71% (95%CI 61-79) (see Table). Subjects entering ASCT in CR had a post ASCT CR rate of 91%, and a 2 year OS rate of 70%. Compared to subjects without CR when entering ASCT, those in CR at time of ASCT were 8-fold more likely to be in CR post ASCT and 3-fold likelihood of being alive at 2 years post ASCT. Difference in survival were not apparent between transplantation in CR1, CR2, or greater than CR2. Achievement of CR appears to be predictive

Meta-analysis: CR and 2 yr OS post ASCT with baseline lymphoma status

	CR post ASCT	2 year OS	Lymphoma Status Entering ASCT				
Rate (%)	70.5	70.9	1st CR = 16%	>CR1 = 8%	PR, CS = 25%	Relapse NOS = 27%	PD/ refractory = 16%
95% Conf. Int.	59.5-79.5	60.9-79.2					

CR = complete remission, PR = partial remission, CS = chemosensitive, PD = progressive disease

of 2-year survival. Summary data regarding histologic subtype, stage, immune status, viral load at ASCT and conditioning regimen will also be presented.
Conclusion: ASCT for HIV(+) patients has a comparable response and survival to the HIV(-) population for ASCT. Consideration of ASCT as an earlier therapeutic option in HIV related lymphoma should be investigated. Additional prospective trials are necessary to determine optimal management of this population.

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DEFINING THE OPTIMAL THRESHOLD OF PERIPHERAL BLOOD (PB) CD34 + CELLS TO INITIATE APHERESIS IN PATIENTS WITH NHL UNDERGOING AUTOLOGOUS HEMATOPOIETIC STEM CELL TRANSPLANTATION (AUTO-HSCT) AFTER G-CSF MOBILIZATION

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Introduction: PB CD34+ cells are routinely monitored to optimize the timing and success of CD34+ stem cell collection after G-CSF ± chemotherapy mobilization in NHL patients (pts) undergoing auto-HSCT. A threshold of ≤10 PB CD34+ cells/μl is often used to predict poor mobilization and aid the decision of whether novel therapies such as plerixafor should be included in the mobilization scheme. This analysis evaluated whether a PB threshold ≤10 cells/μl is indeed the most optimal threshold to predict mobilization failure.
Methods: This was a post-hoc analysis of pts with NHL (enrolled in the phase 3 study 3101) mobilized with G-CSF alone (10 μg/kg SC for up to 8 doses). PB CD34+ cells were measured on the morning of Day 4, ~24H prior to apheresis. The proportion of pts collecting the minimal ($\geq 2 \times 10^6$) or optimal ($\geq 5 \times 10^6$) CD34+ cell dose and apheresis yields were evaluated in pts with PB CD34+ cells/μl ≤10 (PB ≤10) or >10 (PB >10).
Results: This analysis was restricted to patients randomized to receive G-CSF plus placebo. In the 3101 study, 142 pts were mobilized with G-CSF alone; Day 4 PB CD34 cell counts were available for 124 pts. 75/124 (60%) pts had PB ≤10 and 49/124 (40%) pts had PB >10. The median CD34+ cell yield in 2 days was 0.97×10^6 cells/kg in pts with PB ≤10 and 3.3×10^6 cells/kg in pts with PB >10. The median CD34+ cell yield in 4 days was 1.31×10^6 cells/kg in pts with PB ≤10 and 4.52×10^6 cells/kg in pts with PB >10. Only 22.7% pts with PB ≤10 and 65.3% pts with PB >10 collected the minimal cell dose of ≥2 $x10^6$ cells/kg in 2 days. If apheresis was continued for 4 days then 34.7% pts with PB ≤10 and 79.6% pts with PB >10 were able to collect the minimal dose. Similarly, only 5.3% pts with PB ≤10 and 30.6% pts with PB >10 collected the optimal cell dose of ≥5 $x10^6$ cells/kg in 2 days. Continuing apheresis for 4 days allowed 10.7% patients with PB ≤10 and 40.8% patients with PB >10 to collect the optimal dose.
Conclusion: Collectively, these data demonstrate that if apheresis was planned in pts with >10 PB CD34+ cells/μl, then 20.4% pts would not collect the minimal cell dose ($\geq 2 \times 10^6$) and 59.2% pts would not collect the optimal cell dose ($\geq 5 \times 10^6$) in 4 apheresis days. Thus, a threshold of >10 PB CD34+ cells/μl does not ensure adequate stem cell collection in a significant proportion of NHL patients mobilized with G-CSF alone. Higher PB CD34+ thresholds should be evaluated to ensure effective HSC collection in NHL pts proceeding to HSCT.

Mobilization Outcomes in G-CSF Mobilized Patients with PB ≤10 or PB > 10

Median (range) cumulative CD34+ cells/kg x 10^6 after 2 apheresis days	0.97 (0.06 - 9.16)	3.30 (0.46 - 12.00)
Median (range) cumulative CD34+ cells/kg x 10^6 after 4 apheresis days	1.31 (0.06 - 10.58)	4.52 (0.46 - 15.00)
% Pts achieving ≥2 x 10^6 CD34 + Cells/Kg in 2 days	22.7	65.3
% Pts achieving ≥2 x 10^6 CD34 + Cells/Kg in 4 days	34.7	79.6
% Pts achieving ≥5 x 10^6 CD34 + Cells/Kg in 2 days	5.3	30.6
% Pts achieving ≥5 x 10^6 CD34 + Cells/Kg in 4 days	10.7	40.8

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ADDITION OF BORTEZEMIB TO HIGH DOSE MELPHALAN DOES NOT IMPROVE RESPONSE RATE OR PROGRESSION-FREE SURVIVAL

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Background: High dose melphalan remains the preparative regimen of choice for autologous hematopoietic stem cell transplantation (auto HCT) in multiple myeloma (MM). Bortezomib is an active agent in newly diagnosed or relapsed MM, and has synergistic activity with melphalan. We recently conducted two randomized phase II trials in MM, one of which studied the safety and efficacy of adding bortezomib to high-dose melphalan in the preparative regimen for auto HCT. Here we report the response rates, progression-free survival (PFS) and overall survival (OS) between the melphalan-based preparative regimens with or without bortezomib.
Methods: In the first randomized trial 48 patients received high-dose Melphalan at 100 mg/m^2 IV on days -4 and -3, and ascorbic acid (AA) 1000 mg IV daily on days -9 to -3. Patients in arm 1 did not receive arsenic trioxide (ATO); patients in arm 2 received ATO 0.15 mg/kg IV from days -9 to -3; patients in arm 3 received ATO at 0.25 mg/kg from days -9 to -3. In the second randomized trial 60 patients were enrolled and 58 received an auto HCT with a preparative regimen of melphalan 100 mg/m^2 IV on days -4 and -3, AA 1000 mg/day IV on days -9 to -3 and ATO 0.25 mg/kg IV on days -9 to -3. Patients in the second trial were randomized to 3 arms; no bortezomib (arm 1), bortezomib 1 mg/m^2 on days -9, -6 and -3 (arm 2), and bortezomib 1.5 mg/m^2 on days -9, -6 and -3 (arm 3).
Results: A total of 106 patients received an auto HCT in these 2 trials. Of 106 patients, 39 received a preparative regimen with bortezomib (Group A) and 67 without bortezomib (Group B). Patient characteristics and outcome are summarized in Table 1. Patients in group B (without bortezomib) were slightly older, had a longer interval between diagnosis and auto HCT and 21% had a prior auto HCT (vs. 2% in group A). Complete + very good partial response rates (CR + VGPR) in groups A and B were similar (31% vs. 31%). Median PFS and OS in group A vs. B were 14 vs. 23 months ($p = 0.08$) and 80 vs. 82 months ($p = 0.17$), respectively.
Conclusions: In this analysis of 106 patients, adding bortezomib to a HD melphalan preparative regimen did not improve the response rate, PFS or OS.

Patient Characteristics and Outcomes

	Group A (Bortezomib)	Group B (No Bortezomib)
Number of patients	39	67
Median age	59	55
Abnormal cytogenetics	13	11
Relapsed disease	10	25
Prior auto HCT	1	14
Months from diagnosis to auto HCT	9	14
Median CD34 dose 106/kg	3.9	4.56
Median days to engraftment	10	10
Grade 3-4 adverse effects	12	20
CR	7 (18%)	16 (24%)

(*Continued*)